Second impact syndrome in football: new imaging and insights into a rare and devastating condition

Case report

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Premature return to play for the concussed pediatric athlete may result in devastating neurological injury. Identification of at-risk patients and ideal management of the concussed athlete remain challenging for the pediatrician. The authors review a case of second impact syndrome in which neuroimaging was obtained between the first and second impacts, a circumstance which to their knowledge has not been previously reported. This case offers new insights into the underlying pathophysiology of this disease process and potential risk factors for its development.
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KEY WORDS • brain concussion • cerebral concussion • trauma • postconcussion syndrome • brain injury • football

SECOND impact syndrome is a rare, often fatal, traumatic brain injury that occurs when a repeat injury is sustained before symptoms of a previous head injury have resolved.[3] Although many cases have been described in the literature,[2–5,7,8] the existence of SIS remains controversial and the underlying mechanisms remain unclear.[6] We report the first case, to our knowledge, of SIS for which CT of the brain was performed between the first and second impacts, as was early brain MRI following the second impact. This case contributes further evidence of the existence of SIS and provides insight into its underlying pathophysiology.

Case Report

History and Presentation. A previously healthy 17-year-old high school football player sustained a helmet-to-helmet hit with an opposing player during a punt return. He felt momentarily dazed, telling a teammate, "I feel dizzy, I can't really see straight," but continued playing immediately. Witnesses reported that he played the remaining 15 minutes of the game with no apparent difficulty. Following the game, he reported that he had a headache, which he attributed specifically to the hit described, telling a teammate, "That was the hardest I've been hit in my whole life." Over the next 3 days he resumed typical activities but complained of fatigue and persistent headache, exacerbated by lateral upward gaze.

Four days after the game, the patient asked to see his doctor because of continued severe headache. The results of evaluation and neurological examination at his primary care physician's office were normal. (The examining physician specifically noted "coordination and balance good, fundoscopic exam grossly normal, mental status exam normal.") An unenhanced CT scan of the brain was obtained and interpreted as normal at both the initial reading and upon subsequent review by several neurosurgeons and a neuroradiologist (Fig. 1A–C). The patient was advised to refrain from practice until his symptoms resolved; however, he returned to practice that afternoon.

The following day, 5 days after the initial impact, the patient continued to complain about persistent headache and appeared to have difficulty with concentration. At full-dress practice that afternoon, he participated in hitting drills. After a hit during the fourth drill he was slow in getting up. He reported to a teammate that he was "OK

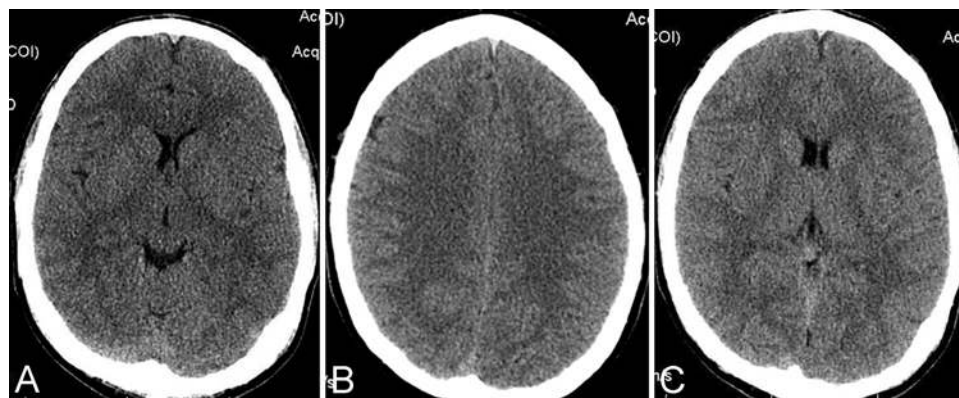*Abbreviations used in this paper:* ICP = intracranial pressure; SIS = second impact syndrome.

FIG. 1. Images obtained after first impact. **A:** Normal head CT scan obtained between first and second impacts. **B and C:** Additional views of the first CT scan showing normal results.

but had a headache." Several plays later he went down on one knee, complained of dizziness and headache, and said he could not feel his legs. He subsequently became unresponsive, and there was generalized seizure activity. He was intubated at a local emergency department where noncontrast CT of the brain demonstrated thin bilateral subdural hematomas. He was treated with lidocaine, mannitol, fosphenytoin, fentanyl, and midazolam prior to and during aeromedical transport to our institution, a tertiary trauma and neurosurgical center.

Examination. In our facility, the patient was afebrile; his blood pressure was 143/79 mm Hg and heart rate was 93 bpm. Neurological examination, conducted by a neurosurgeon, revealed a Glasgow Coma Scale score of 7T ("T" indicating intubation) with eye opening to noxious stimuli, bilateral withdrawal of upper extremities, and mixed posturing of his legs. Pupils were 3 mm bilaterally and sluggish. He had Grade 3+ reflexes throughout, bilateral clonus, and an absent Babinski reflex. Repeat noncontrast CT of the brain (Fig. 2A) demonstrated subdural hematomas and mild cerebral swelling. Coagulation studies and chemistry panel were normal. Urine toxicology screen was negative. An ICP monitor demonstrated initial pressures ranging from 25–30 mm Hg (normal range 5–15 mm Hg), and MRI of the brain and cervical spine performed during the initial 18 hours revealed mild downward transtentorial herniation, bilateral subdural hematomas, and abnormal T2 signal and restricted diffusion in the medial left thalamus (Fig. 2B and C). Midline structures, including the thalamus and hypothalamus, were displaced caudally. There was no evidence of vascular or upper cervical spinal cord injury and no evidence of white matter shear injury or cerebral edema.

Treatment and Outcome. Despite optimizing sedation his ICP remained elevated, requiring management with mannitol, 3% normal saline, an external ventricular drain, and induction of a pentobarbital coma. Noncontrast CT of the brain, repeated on his 3rd hospital day, revealed stable bilateral subdural hematomas, 4 mm of right-to-left subfalcine shift, and effacement of the frontal and occipital horns. Subsequent imaging demonstrated resolution of his subdural hematomas and areas of encephalomalacia, including both thalami, the medial frontal lobes, and the

splenium of the corpus callosum (Fig. 3A–C). His hospital course was complicated by hypotension, which required vasopressor therapy with norepinephrine, epinephrine, and dopamine; severe metabolic acidosis and renal failure requiring hemodialysis; sepsis; ventilator-associated pneumonia with empyema requiring thoracotomy; disseminated intravascular coagulation; and cardiac arrest. He was transferred to rehabilitation on hospital Day 54, and discharged to home on hospital Day 98. At the time of discharge he was nonverbal and nonambulatory. Today, more than 3 years after his injury, our patient is living at
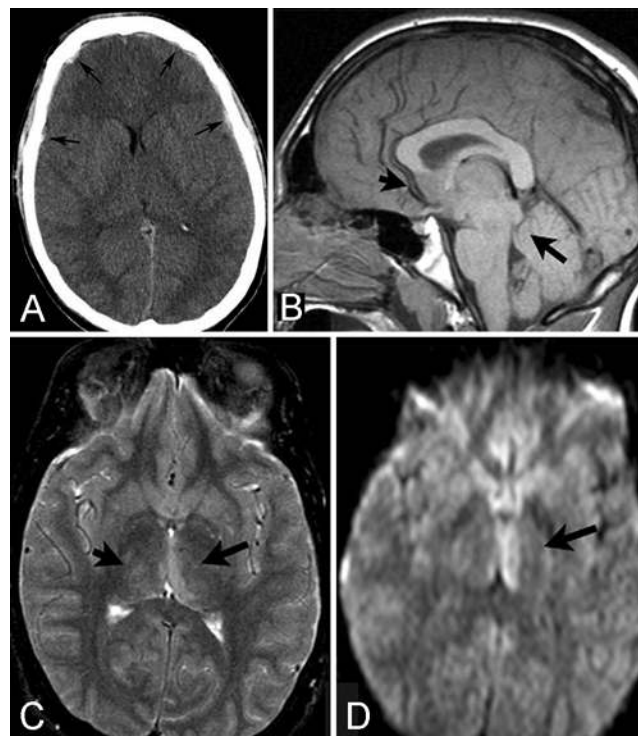

FIG. 2. Images obtained after second impact. **A:** Head CT obtained after second impact. *Arrows* point to thin bilateral subdural hematomas. **B:** Sagittal T1-weighted brain MR image. *Arrows* point to downward descent of the midline structures. **C:** Axial T2-weighted MR image. *Arrows* point to thalamic injury. **D:** Axial diffusion-weighted MR image. *Arrow* points to left thalamic injury. Restricted diffusion was proven by calculation of apparent diffusion coefficient (not shown).
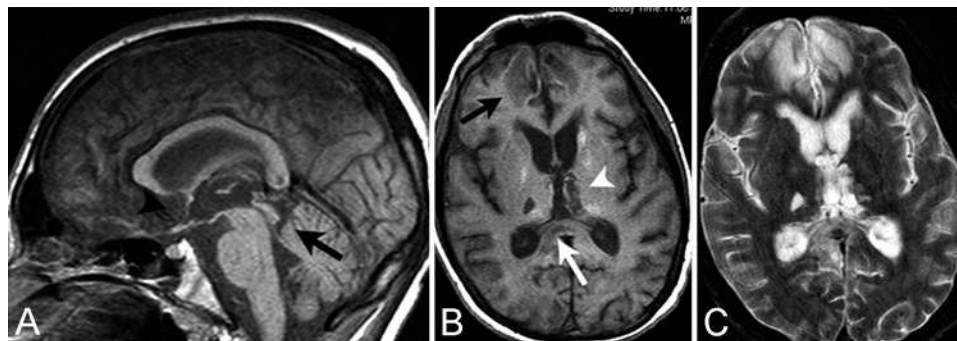
FIG. 3. Magnetic resonance images obtained 2 months after second impact. **A:** Sagittal T1-weighted image. *Arrow* indicates resolution of downward descent of midline structures seen in previous MR study (compare with Fig. 2B). **B:** Axial T1-weighted image. *Black arrow* points to injury to the medial frontal lobes. *Arrowhead* points to left thalamic injury matching the abnormality seen acutely on diffusion imaging (compare with Fig. 2C). *White arrow* points to injury to the splenium of the corpus callosum. **C:** Axial T2-weighted image demonstrating the same injuries.

home and has regained limited verbal, motor, and cognitive skills.

Discussion

The pathophysiology of SIS is poorly understood, but has previously been thought to reflect loss of cerebral autoregulation and resultant massive brain swelling, frequently with brain herniation. A number of authors have hypothesized that this disruption of autoregulation results from reinjury to neuronal cells within a vulnerable period of ongoing derangement from previous injury.[2,6,11] In the original description of SIS it was suggested that the derangement following the first impact involves a space-occupying injury, probably evident on CT, and that a CT examination of the brain may be indicated before return to play is allowed for some athletes.[11] Our case, while supporting the existence of this clinical entity, disproves this hypothesis, suggesting instead that the initial impact results in derangement at a level not visible on routine CT scanning. We are well aware that there can be axonal injuries, small extraaxial hematomas, and punctate hemorrhages visible on MRI in patients with a "normal" CT scan. However, brain compression by an extraaxial mass did not play a role in the symptoms of our patient, and even the MR scan done soon after admission following his second impact showed only a very thin subdural hemorrhage. Importantly, this case shows that a normal head CT scan does not obviate the need for close clinical follow-up and for the athlete to be cognitively normal and asymptomatic before return to play.

In addition, our imaging supports a previously postulated model of brain injury in which cerebral blood flow dysautoregulation results in massive hyperemia.[1,5] In this model, profound rapid blood flow dysautoregulation and vascular engorgement may result in fatal or near-fatal hyperemic herniation of the brain.[1,3,5] Upon arrival at our facility, CT of our patient's brain demonstrated minor subdural hematomas, and no evidence of vasogenic edema was seen on subsequent brain MRI, yet his opening intracranial pressures were 25–30 mm Hg. It is our opinion that his structural brain injuries were insufficient to explain this elevation in intracranial pressures. Brain MRI demonstrated caudal displacement of midline structures, with injury to both thalami resulting from transtentorial herniation. We believe that these findings represent pressure effect from a hyperemic brain. Subsequent brain imaging demonstrated resolution of this displacement as well as evidence of tonsillar injury consistent with herniation.

Return-to-play guidelines for concussion and head injury in football continue to evolve, with a greater emphasis on computerized neurocognitive testing in recent years. However, distinguishing the transient "stun" that many football players experience after a hit from that of a concussion is challenging and identification of at-risk players is difficult. One of the most alarming features of our case is that our patient suffered what has been historically euphemized as just a "ding" during a play that was not extraordinary in any way. Outwardly he did not appear to require removal from play. Of interest, our patient's primary complaint prior to his second impact was severe headache, but headache is a common manifestation of concussion. In a study of prevalence of headaches in high school and collegiate football players, 85% of athletes reported headache related to being hit during football. Notably, few athletes reported their symptoms to trainers or coaches. Many reported playing with a headache, but none of them subsequently suffered from SIS.[10]

It remains unclear why one player suffers from SIS while another does not, or why this syndrome seems to affect athletes in one sport over another. Review of the case reports clearly suggests that younger players are at greater risk than older players, and this finding may be related to differences in the physiology of the pediatric brain.[1,6] In fact, virtually all of the reported cases of SIS have occurred in high school athletes and adolescents. No cases have been reported in National Football League players.[9] We believe that the clinical features of this case may help us identify patients at increased risk of SIS. Specifically, the persistence, duration, and severity of our patient's headache after the initial impact suggest significant ongoing neurophysiological pathology despite normal head CT. In previously reported cases of catastrophic brain injury from suspected SIS, profound and persistent headache has been frequently noted.[2,3] Included in these series are cases like ours in which an athlete, invested in

continuing to play, independently sought care for intractable symptoms. This characteristic complaint suggests that profound ongoing headache may be a more specific predictor than other subjective symptoms. We consider the possibility that this severe headache may reflect mild intracranial hypertension from hyperemia following the first insult. Furthermore, the rarity of this syndrome and the likelihood that a significant number of concussed athletes have played while symptomatic may be indicative of a population subset that is still unidentified. At this time the definitive etiology remains elusive.

Given the conservative estimate of 300,000 sports-related traumatic brain injuries annually in the US,[4] decisions made regarding return to play should fully take into consideration the best evidence about subsequent health risks. This must include the rare occurrence of SIS and sudden death. As in this case, competitive athletes will frequently desire to return to play before they are medically fit to do so and may not be forthcoming about their true physical condition. Although an increasing number of high school teams have athletic trainers and team physicians, many do not. It is therefore imperative that physicians familiar with sports concussions take the lead in educating coaches, athletes, families, and primary care and emergency physicians about sports-related head injuries and their potential risks and consequences.

Disclosure

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